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Effect of Coconut Oil Administration on Some Hemostatic Changes Associated with Obesity in Rats

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Abstract

In the last few decades, coconut oil was claimed to have some beneficial health effects, attributed mainly to its medium chain triglycerides. It was, thus, intriguing to investigate the potential benefit of coconut oil in alleviating the prothrombotic tendency often encountered in obese individuals. The present study was carried out on 44 rats, of both sexes, aged 10 days at the start of the study. 31 out of 44 rats were offered high caloric diet (the cafeteria diet) for induction of obesity. Rats were allocated into the following groups: **Group1: Control rats (C) (**n=13 rats), comprising rats fed on the standard chow diet all-over the study period (24 weeks). **Group2: Cafeteria diet-fed rats (Caf)** (n=16 rats), comprising rats fed on cafeteria diet until the end of the study period and **Group3: Cafeteria diet/coconut oil-fed rats** (**Caf/Coco**) (n=15 rats), comprising rats fed on cafeteria diet with coconut oil starting from the 16th week till the end of the study period. At the end of the study, the BMI was assessed in the 3 studied groups and blood samples were collected for determination of platelet count and aggregation, prothrombin time (PT), activated partial thromboplastin time (APTT), fibrin degradation products (FDPs), and plasma lipid profile. The encountered results revealed that the mean BMI of the cafeteria diet-fed rats was significantly higher than the BMI of control rats, and that the mean BMI of rats receiving cafeteria/coconut oil diet for 9 weeks was significantly decreased compared to their matched caf group. The PT, APTT and platelet count were all non significantly different in the three studied groups. Platelet aggregation, on the other hand, was significantly increased in the caf group compared to the control group, and significantly decreased in the caf/coco group compared to both the caf group and the control group. The plasma FDPs levels were not significantly different in the 3 studied groups. The lipid profile was insignificantly different in the 3 studied groups except in the caf/coco group which revealed a significant elevation of total cholesterol and HDL-c compared to caf group. The present findings, thus, point to the possible beneficial effect of coconut oil feeding on obesity - induced enhanced platelet aggregation.

{* From M.Sc. Thesis, Physiology department}

Introduction

The prevalence of obesity is increasing worldwide, although the proportion varies from country to country and between geographical areas within a country ***(WHO, 1998).*** Obesity is known to predispose to a number of cardiovascular risk factors, including hypertension, elevated cholesterol and impaired glucose tolerance ***(WHO report, 2000)***. Also, Obese individuals are known to be susceptible to thrombotic diseases, though the underlying mechanism is still unknown ***(Yamamoto et al., 2005)***.

In many areas of Sri Lanka, the coconut tree and its products have for centuries been an integral part of life, and it has come to be called the "Tree of life". However, in the last few decades, the relationship between coconut fats and health has been the subject of much debate. Around 92% of these fats are saturated fats, which led to the belief that coconut fats are 'bad for health', particularly in relation to ischemic heart disease. Yet, since most of the saturated fats in coconut oil are medium

chain fatty acids whose properties and metabolism are different from long chain triglycerides (LCT), they are now believed to be not as 'bad for health' as other saturated fats ***(Amarasiri and Dissanayake, 2006).*** In fact, the medium-chain fatty acids and monoglycerides found primarily in coconut oil were found to have miraculous healing power ***(Kabara, 2000)***. The author added that it is rare in the history of medicine to find substances that have such useful properties and still be without toxicity or even harmful side effects**.**

A link between coconut oil and obesity has been postulated. ***Thampan (1994)*** stated that any health condition is made worse if the metabolic rate is slower than normal because cells cannot heal and repair themselves as quickly. The author added that increasing metabolic rate, therefore, provides an increased degree of protection from both degenerative and infectious illnesses. Medium chain fats present in coconut oil were found to stimulate thermogenesis to a greater degree than does excess energy intake as LCT) (***Binnert et al., 1998)***. Further, excess energy derived from medium chain triglycerides (MCT) was reported to be stored with a lesser efficiency than is excess energy derived from dietary LCT ***(Hill et al., 1989 and Noguchi et al., 2002),*** resulting in greater loss of adipose tissue. Thus, MCT might be considered as agents that aid the prevention of obesity or potentially stimulating weight loss ***(St-Onge et al., 2003).***

It was, thus, intriguing to investigate the effect of coconut oil feeding on the hemostatic changes associated with obesity, aiming at alleviating the prothrombotic tendency often encountered in obese individuals.

Materials and Methods:

Animals:

The present study was carried out on 44 rats, of both sexes, aged 10 days at the start of the study ***(Rodriguez et al., 2001$_a$)***. Rats were purchased from the Institute of Ophthalmology in Giza, and maintained in the Physiology Department animal house under standard conditions of boarding.

NB: Rats were separated into male and female cages at the age of 40 days to prevent mating.

Experimental protocol:

Rats included in the present study were divided into 2 main categories: control rats and rats rendered obese by being fed high caloric diet (the cafeteria diet). All animals received standard rat chow in the first week for acclimatization to the new facilities, the cafeteria diet being started on the second week.

Rats were allocated into the following groups:

- **Group1: Control rats (C) (**n=13 rats), comprising rats fed on the standard chow diet all-over the study period (24 weeks).
- **Group2: Cafeteria diet-fed rats (Caf)** (n=16 rats), comprising rats fed on cafeteria diet, for induction of obesity till the end of the study period.
- **Group3: Cafeteria diet/coconut oil-fed rats** (**Caf/Coco**) (n=15 rats), comprising rats fed on cafeteria diet for 15 weeks, thereafter they were fed cafeteria diet mixed with coconut oil (cafeteria/ coconut oil diet) till the end of the study period.

The diet formulae supplied to the three studied groups were as follows:

A)Standard chow diet

All rats in the period of acclimatization were fed on the formula AIN-93G ***(Reeves et al., 1993).*** Control rats were maintained on this formula till the age of 3 months. From the age of 3 months till the end of the study, control rats were fed on the formula AIN-93M ***(Reeves et al., 1993)*** which provided 18% of energy as protein, 76% of energy as carbohydrate, and 6% of energy as lipids, by dry weight.

B) The cafeteria diet

This diet was composed of the following items: patè, chips, chocolate, bacon, biscuits and standard chow diet, in a proportion of 2:1:1:1:1:1 (***Berraondo et al., 2000).*** The 6 food items were crushed to a powder form and mixed thoroughly. The powder was then placed in a container and given to the animals.

C) The modified cafeteria diet with coconut oil

This diet consisted of a mixture of cafeteria diet and coconut oil (purchased from Morgan factory) in a ratio of 75 gm cafeteria diet and 25gm coconut oil ***(Zulet et al., 1999).*** This diet was supplied to rats in group 3 (Caf/Coco) for 9 weeks.

The three food formulae adopted in the present study were analysed in the National Nutrition Institute (NNI), according to AOAC 2003, to confirm their conformity to the cited diet characteristics. The energy provided by each diet formula, and by each of its different components, was found to be as follows:

Diet	Energy / 100 gm diet	% of energy as CHO	% of energy as protein	% of energy as fat
Control diet	**418.98**	**71.19**	**15.28**	**13.53**
Cafeteria diet	**571.54**	**32.76**	**8.19**	**59.05**
Cafeteria/Coconut oil diet	**642.1**	**24.44**	**5.34**	**70.22**

The served amount of food was 10gm/rat at the start, and was increased gradually over the whole study period, reaching 25 gm/rat at the end of the study. Every day, the amount of food remaining from the previous day was weighed to monitor the daily food consumption.

The body weight of rats in the different groups was assessed weekly to monitor the progress in weight gain of each group.

Experimental Procedures:

On the day of sacrifice, overnight fasted rats were weighed and anesthetized by intraperitoneal injection of thiopental sodium (40 mg/Kg, b.w.), then the body height was measured. A midline abdominal incision was made. The abdominal aorta was exposed and cannulated with a catheter and three blood samples were collected into 3 plastic tubes, one of which contained 3.2% trisodium citrate (9 volumes of blood + 1 volume citrate), the 2nd tube contained a drop of heparin, and the 3rd tube contained EDTA.

The citrated blood sample was centrifuged at 1000 rpm for 5 min., platelet rich plasma (PRP) was pippetted off, and kept in a plastic tube for assessment of platelet aggregation. The rest of the sample was centrifuged at 3000 rpm for 15 min. for separation of platelet poor plasma (PPP). A part of the PPP was used in the assessment of platelet aggregation as described by ***Born (1962)*** and ***O'Brien (1963)***, and the rest kept in a plastic tube at room temperature for performance of the following coagulation tests (within 4-6 hours) :

1-Prothrombin time (PT), as described by ***Neofotistos et al. (1998),*** using kits (Neoplastine CI Plus) supplied by Diagnostica Stago.

2-Activated partial thromboplastin time (APTT), as described by ***Contant et al. (1983),*** using kits (C.K.Prest) supplied by Diagnostica Stago.

3-An aliquot of the PPP was stored frozen for determination of fibrin degradation products (FDPs), as described by ***Mirshahi et al. (1986),*** using kits supplied by Diagnostica Stago.

The heparinized blood sample was centrifuged at 3000 rpm for 15 min. and the separated plasma was used for determination of plasma levels of:

1-Triglycerides, as described by ***Fossati and Prencipe (1982),*** using kits supplied by Biolabo SA.

2-Total cholesterol, as described by ***Allian et al. (1974)***, using kits (CHOD-PAP) supplied by Greiner Diagnostic Gmbh.

3-HDL-C, as described by ***Lopes-Virella (1977)***, using kits supplied by Greiner Diagnostic Gmbh**.**

LDL-C, was calculated as follows: LDL-C = (TC) – (HDL-C + TG/5) ***(Friedewald et al., 1972).***

The blood sample taken on EDTA was used for determination of platelet count, using coulter T- 660, depending upon electronic counting, according to the method described by ***Coulter (1956)***.

The body mass index (BMI) was calculated as follows: ***BMI = body weight in gm / square height in cm²*.**

The height of rats was measured as the distance (in cm) between the nose and the anus, using an ordinary ruler. The body weight (in gm) was determined by the use of the ordinary animal scale.

Statistical analysis

All statistical data and significance tests were performed by using SPSS (Statistical Program for Social Science) statistical package (SPSS Inc) version 10.0 ***(Armitage P and Berry G, 1987***). Statistical significance was determined by one-way ANOVA (analysis of variance) for differences between means of different groups; further analysis was made by LSD (least significance difference) multiple-range test to find intergroupal differences; a probability of $P< 0.05$ was considered statistically significant. Chi-square test was used for comparison of qualitative variables.

RESULTS:

Changes in body weight and BMI in the different studied groups:

Figure (1) shows the progressive increase in body weight in the three studied groups, assessed weekly, throughout the study period up to the 8th week, the mean body weight of the cafeteria diet-fed rats was significantly lower than control rats. Starting from the 11th week to the 15th week, the mean body weight of cafeteria diet-fed rats was higher compared to the control rats.

After the 15th week, cafeteria diet-fed rats were subdivided into cafeteria diet fed group (caf) and cafeteria/coconut oil diet fed group (caf/coco). Mean body weights of the three studied groups were comparable from 16th to the 21st week. Then, mean body weight of the caf/coco group was significantly lower than control and caf groups at the 23th week and from caf group at the 22th and 24th weeks.

As regards the BMI, which was calculated at the end of the study period, it was found that the mean BMI of the cafeteria diet-fed rats was significantly higher than the mean BMI of control rats ($P<0.02$). On the other hand, the mean BMI of caf/coco oil fed rats was significantly decreased compared to their matched cafeteria diet-fed rats ($P<0.01$). However, it was insignificantly different from control group (table 1 and figure 2).

Fig. (1) The mean body weights (gm), assessed weekly, of control, cafeteria diet-fed and cafeteria/coconut oil diet-fed rats throughout the period of the study.

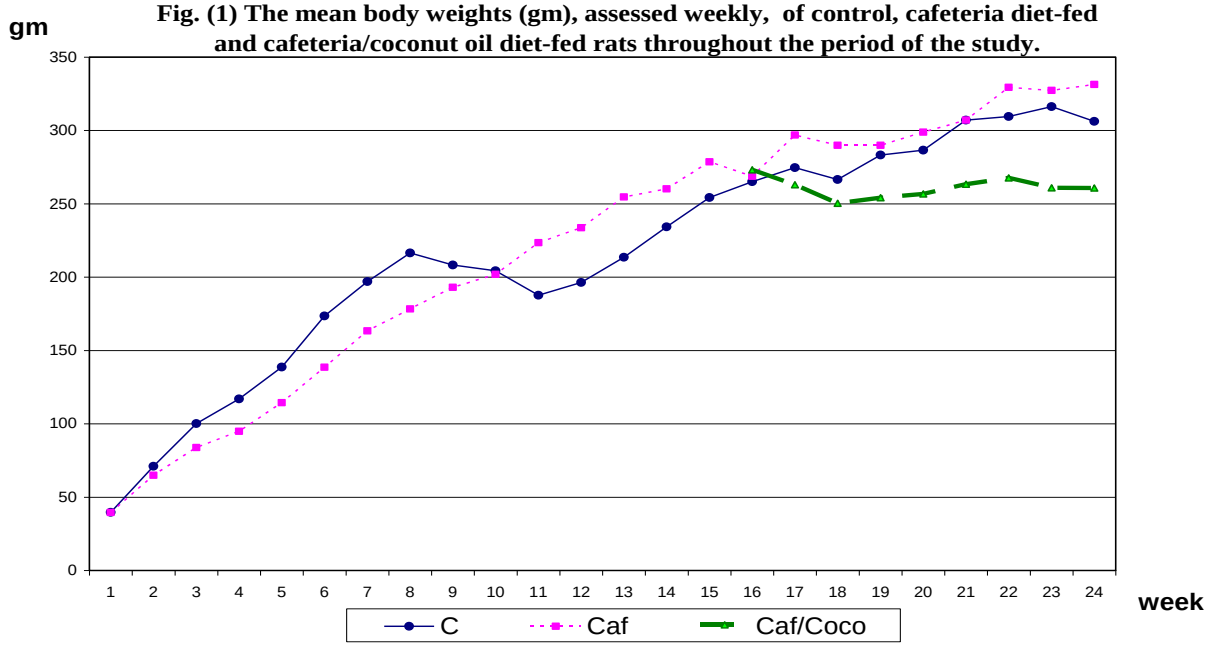
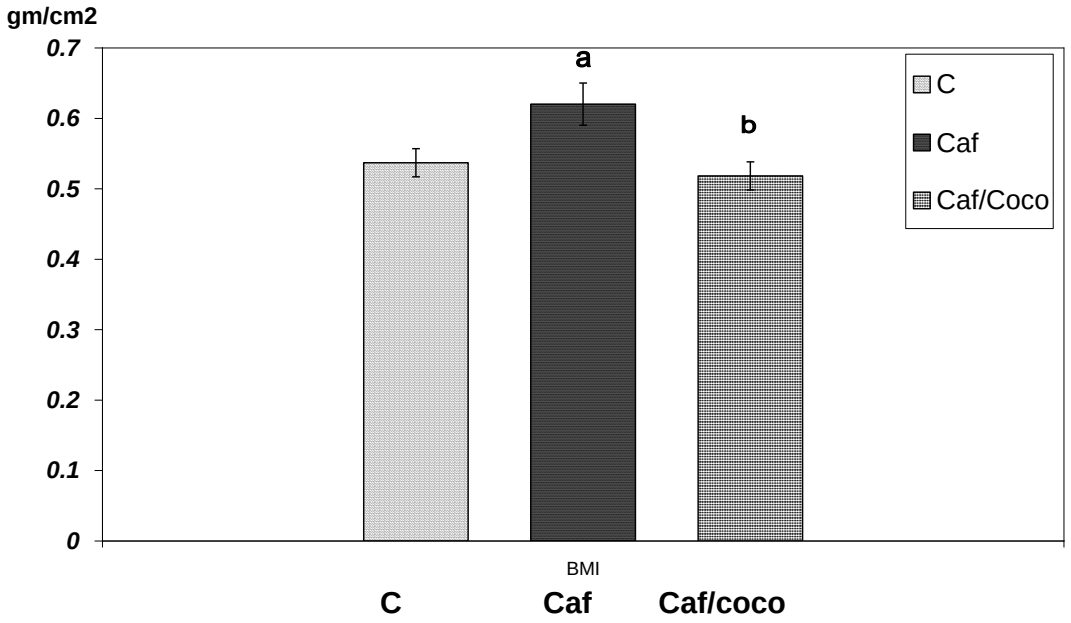


Fig. (2): Changes in BMI (gm/cm²) in Control (C), Cafeteria diet-fed (Caf) and Cafeteria/Coconut oil diet-fed (Caf/coco) groups.



a = Significant difference from the control group, calculated by LSD at P < 0.05 for unpaired data.

b = Significant difference from the cafeteria/coconut oil diet-fed group, calculated by LSD at P < 0.05 for unpaired data.

Changes in the Hemostatic parameters:

Prothrombin time (PT), partial thromboplastin time (PTT) and platelet count were all non-significantly different in the three studied groups. Table (1); Figure (3).

Platelet aggregation, on the other hand, was significantly increased in the cafeteria diet-fed group compared to the control group ($P<0.05$), and significantly decreased in the cafeteria/coconut oil diet-fed group compared to the cafeteria diet-fed group ($P<0.001$) as well as the control group ($P<0.01$) (table 1 and figures 4 & 5).

As regard the plasma **fibrin degradation products (FDPs)**, although the levels were not significantly different in the 3 studied groups, yet the number of observations of the higher levels (>5<20 and ≥20 µg/ml) was greater in the cafeteria diet-fed and cafeteria/coconut oil diet-fed groups compared to the control group (table 2).

Changes in Plasma Lipid Profile

Total cholesterol and HDL were significantly elevated in caf/coco oil fed rats compared to cafeteria diet fed rats ($P < 0.01$ & $P<0.05$ respectively). However, plasma TG and LDL-C levels were comparable in the three studied groups (table 1).

Table 1: Body mass index (BMI, gm/cm²), Prothrombin time (PT, sec), Partial thromboplastin time (PTT, sec), Platelet number (Pl. no., x10³/mm³), Platelet aggregation (Pl. agg., %), Triglycerides (TG, mg/dl), Total Cholesterol (TC, mg/dl), High density lipoproteins cholesterol (HDL-C, mg/dl) and Low density lipoproteins cholesterol (LDL-C, mg/dl) in the 3 studied groups.

Group	BMI (gm/ cm²)	PT (sec.)	PTT (sec.)	Pl. No. (x10³/ Mm³)	Pl. Agg. (%)	TGs (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)
C (n=13)							(n=11)		(n=11)
Mean	0.54	17.85	19.62	740.45	64.89	57.16	75.7	40.55	23.61
±SEM	±0.02	±1.68	±1.43	±37.64	±2.03	±4.51	±3.5	±3.63	±2.28
Caf (n=16)							(n=12)		(n=12)
Mean	0.63	16.63	20.94	622.38	72.77	50.93	67.95	36.64	23. 6
±SEM	±0.03	±1.28	±1.31	±75.88	±2.79	±4.10	±2.4	±3.11	±3.4
P1	<0.02	NS	NS	NS	< 0.05	NS	NS	NS	NS
Caf/Coco (n=15)							(n=10)		(n=10)
Mean	0.52	15.93	18.07	711.93	53.57	54.80	83.3	46.54	24.1
±SEM	±0.02	±0.72	±1.42	±68.19	±2.47	±6.33	±2.7	±2.83	±3.5
P1	NS	NS	NS	NS	< 0.01	NS	NS	NS	NS
P2	<0.01	NS	NS	NS	< 0.01	NS	< 0.01	< 0.05	NS
P3	<0.01	NS	NS	NS	<0.01	NS	<0.02	NS	NS

P1 : Significance of difference from control rats, calculated by LSD at $P < 0.05$ for unpaired data

P2 : Significance of difference from untreated cafeteria fed diet rats calculated by LSD at $P < 0.05$ for unpaired data

P3: Significance by 1-way ANOVA among the 3 studied groups

NS : No significant difference

Table (2):Plasma FDPs in the 3 studied groups

Group		<5 µg/ml	5-20 µg/ml	>20 µg/ml	P1	P2	P3
C (n=13)	Numb. Of rats	9	1	3			NS
	%	69.3%	7.7%	23%			
Caf (n=16)	Numb. Of rats	7	5	4	NS		
	%	43.75%	31.25%	25%			
Caf/Coco (n=15)	Numb. Of rats	6	3	6	NS	NS	
	%	40%	20%	40%			

P1: Significance of difference compared to C group, calculated by chi square test.

P2: Significance of difference compared to Caf group, calculated by chi square test.

P3: Significance of difference between the 3 groups, calculated by chi square test.
NS : No significant difference

Fig (3): Mean changes in Prothrombin time (PT, sec.) and Partial thromb-oplastin time (PTT, sec.), Platelet count (plat co, /1000) in Control (C), Cafeteria diet-fed (Caf) and Cafeteria/coconut oil diet-fed (Caf/Coco) rats.

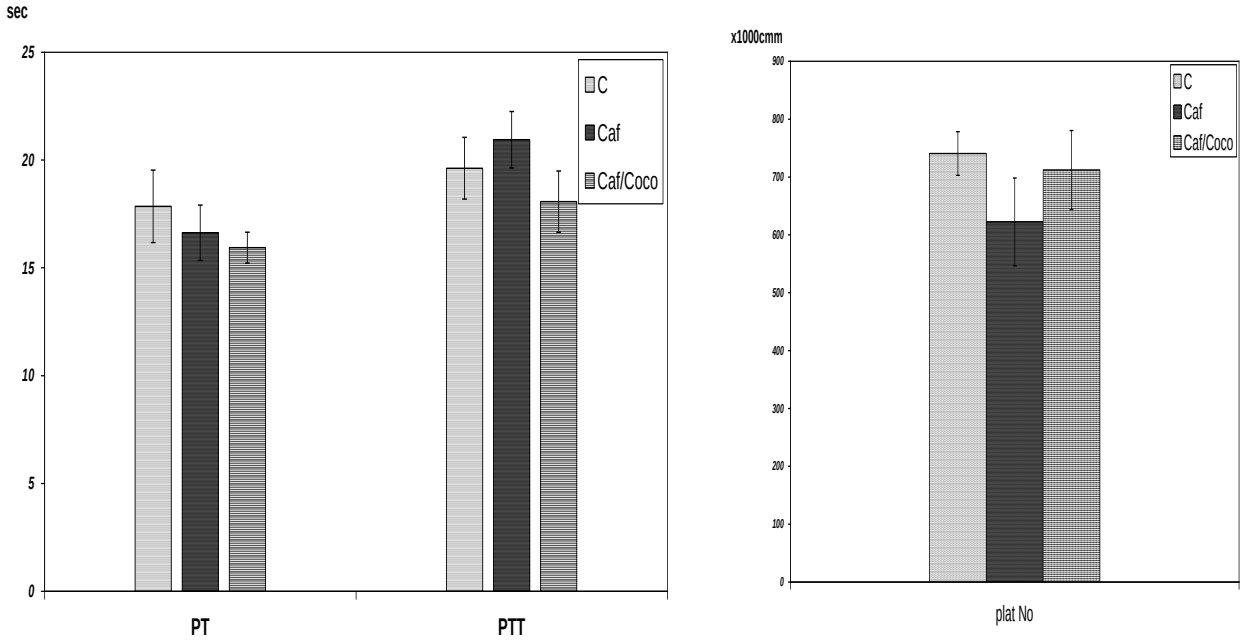
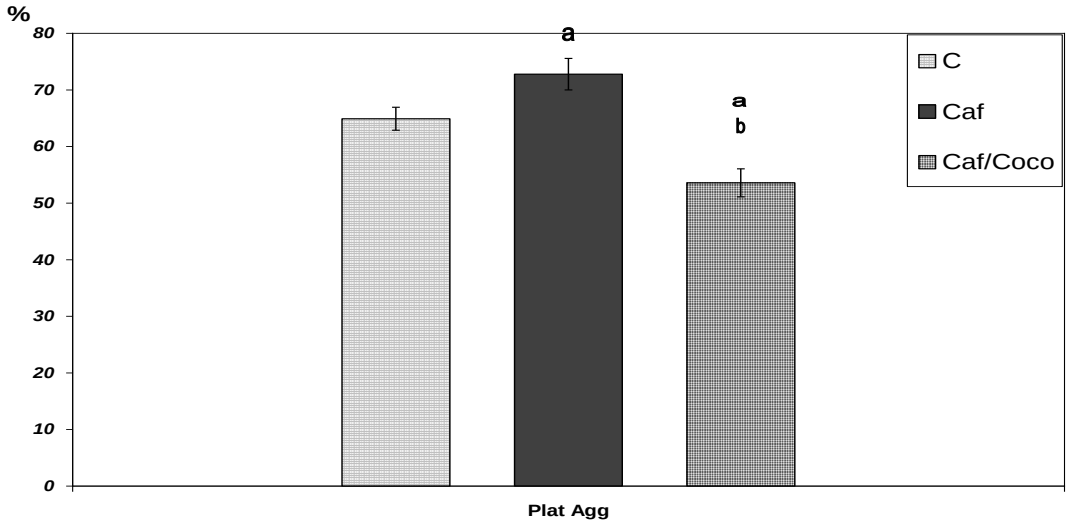
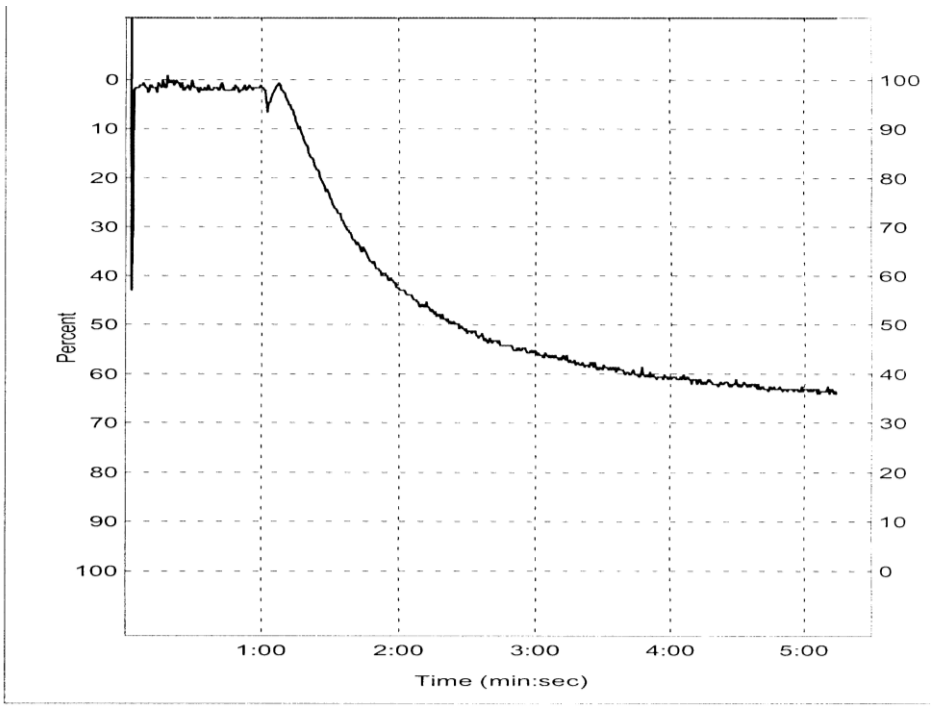


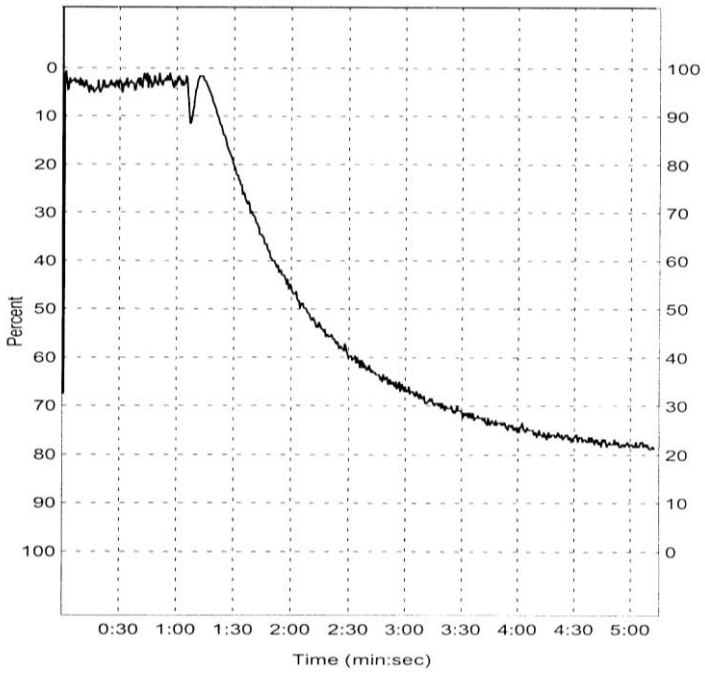
Fig.(4): Mean changes in platelet aggregation (%) in Control (C),Cafeteria diet-fed (Caf) and Cafeteria/coconut oil diet-fed (Caf/Coco) rats.

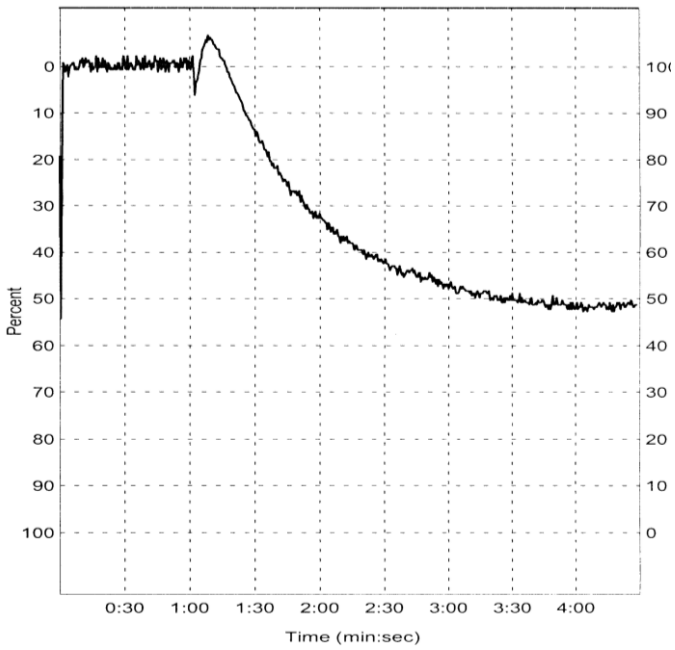


a = significant difference from the control group, calculated by LSD at P < 0.05 for unpaired data.
b = significant difference from the cafeteria/coconut oil diet-fed group, calculated by LSD at P < 0.05 for unpaired data.

(a)







(b) (c)

Fig.(5): Platelet aggregation tracings of control (a), cafeteria diet-fed (b), and cafeteria/coconut oil diet-fed rats (c)

Discussion

The present study was carried out on a rat model of obesity, started in early life. Obesity was induced by feeding suckling rats a high caloric diet (the cafeteria diet). This diet was reported to be a palatable hypercaloric and hyperlipidic diet that can induce voluntary hyperphagia and fast body weight gain **(*Lowell et al., 2000 and Rodriguez et al., 2001b)*.**

It is worth-mentioning that the increase in body weight of the cafeteria diet–fed rats (Caf) showed slowness at the beginning compared to the control group, the mean body weight being lower than that of the control rats. Thereafter, there was a catch up period from the ninth week on, where the weight gain of the cafeteria diet–fed rats became faster, and the mean body weight became greater than the mean body weight of the control group till the end of the experimental period. The initially observed slower weight gain in the cafeteria diet–fed rats may be explained by reduced food intake observed at the beginning of the period of the study. This initial decrease in food intake could have resulted from one or more of the many causes reported to contribute to decreased food intake, e.g. diet palatability, high energy content of the diet ***(Menaker and Navia, 1973),*** low protein content of the diet ***(Rothwell and Stock, 1988),*** and the high fat content of the diet, which has been reported to stimulate the release of cholycystokinin which decreases feeding mainly by activation of the melanocortin pathway of the hypothalamus ***(Guyton & Hall, 2006).*** The lack of marked weight gain in the cafeteria diet-fed rats is likely to be due to diet-induced thermogenesis (DIT), reported to be caused by increased fat content of the diet **(Matsuda *et al., 1997)***, or increased energy content of the diet ***(Rothwell and Stock, 1986)***, both of which are present in cafeteria diet. ***Rothwell and Stock (1982)*** explained cafeteria-diet induced thermogenesis by hypertrophy and hyperplasia of brown adipose tissue. Later on, ***Christoffolete and Moriscot (2004)*** explained DIT in rats fed cafeteria diet by increased total brown fat mitochondria, uncoupling protein percentage and total brown fat uncoupling protein.

Although weight gain of the cafeteria diet-fed rats was not marked, yet there was significant increase in BMI in this group, compared to the control group, indicating increased body adiposity ***(Guyton & Hall, 2006)***. The higher adiposity with cafeteria diet feeding was, also, reported by ***Rodrígue et al. (2004) and Matute et al. (2007***). Increased adiposity in response to cafeteria diet feeding was reported to be related more to an increase in the amount of visceral fat rather than an increase in subcutaneous depot (***Rodrígue et al., 2004).*** The significant increase in BMI in the cafeteria diet-fed rats, without parallel marked overweight could be explained by reduced rate of increase in body length, observed in this group compared to the control group, resulting in a higher ratio between body weight and body length. The reduced rate of body length increase could be a manifestation of reduced growth hormone (GH) secretion in cafeteria-diet fed rats. Decreased GH secretion in cafeteria-diet fed rats was reported by ***DeSchepper et al. (1998)*** as well as by ***Zhou et al. (1998).*** The later demonstrated lower GH release from normal pituitary cells incubated in serum from overfed rats than after incubation with serum from non obese rats.

Addition of coconut oil to the cafeteria diet resulted in significant decrease in body weight of rats after 9 weeks, despite the high energy content of the diet. BMI was also significantly decreased, becoming even lower than the control value. Food intake of rats in this group was observed to be comparable to that of the cafeteria diet fed rats and control rats, which make increased energy expenditure induced by coconut oil feeding in these rats is the most likely explanation of the significant decrease in their body weights and BMI.

Weight lowering effect of coconut oil has been reported in previous literature

(Binnert et al., 1998 and St-Onge et al., 2003), and was attributed to its high content of medium-chain triglycerides (MCT). MCT were found to be digested very quickly and used for energy production, much like carbohydrates, and, therefore, they do not circulate in the bloodstream like other fats, and they do not supply fat to fat cells or contribute to weight gain (***Crozier et al.,* 1987**). Studies have shown that MCT in coconut oil boost metabolism, and increase fatty acid oxidation (***St-Onge et al., 2003)***, thus contributing to decreased body weight and BMI (***Tsuji et al., 2001*).**

As regards the hemostatic mechanisms, the changes in both PT and APTT in the caf and caf/coco groups were statistically non-significant compared to the control group. However, though platelet count was not significantly different in the 3 studied groups, yet platelet aggregation was significantly enhanced in the caf group compared to the control group, and was significantly decreased in the Caf/coco group compared to both the control and the caf groups. Plasma FDPs level was comparable in the 3 studied groups.

The significant enhancement in platelet aggregation observed in the cafeteria diet-fed rats, despite insignificant change in platelet count point to altered platelet function. The impact of obesity on platelet aggregation has long been recognized in both human and animal studies. ***Sonhee et al* .(2004)** reported that platelet aggregation was enhanced and tended to have shorter lag time in obese males compared to non obese males. Also, ***Anfossi et al. (2004***) found that central obesity induced platelet resistance to the antiaggregating effects of prostacyclin and NO, due to impaired cyclic nucleotide synthesis and action; the main effectors of platelet antiaggregation. The authors added that this accounts for platelet hyperactivity in obesity**.** Also, platelets from obese individuals were found to express leptin receptors, which mediated enhanced platelet aggregation to ADP after pretreatment with leptin ***(Corsonello et al., 2002)***.

Platelet aggregation was found to be significantly decreased in coconut/cafeteria diet -fed rats compared to cafeteria diet-fed rats as well as control rats. This favorable effect could be due to decreased body adiposity and decreased leptin level which was reported to be involved in increased platelet activation in obese individuals. This finding disagree with the findings of ***Podbielski et al. (1989)*** and ***Pronczuk et al. (1991)*** who reported increased platelet factor 4 and platelet activation with coconut oil feeding.

Concerning the plasma lipid profile, the encountered results disagree with of earlier reports ***of Pagliassotti et al. (1996) and Anurag and Anuradha (2002)*** that high calorie diet- feeding and obesity result in dyslipidemia.. Lack of dyslipidemia in the present study may be explained by the early start of the high calorie diet (3^{rd} week of rats' lives). ***Serisier et al. (2008)*** reported that younger animals were better able to balance energy needs with energy consumption and added that young animals didn't exhibit significant changes in triglycerides and free fatty acid concentrations compared to older animals.

When coconut oil was added to the diet, a significant increase in total cholesterol and HDL-cholesterol, compared to the caf group, was observed. These findings disagree with those of ***Kaunitz and Dayrit (1992), and Kasai et al. (2003),*** who reported hypocholesterolemic effect of coconut oil. This difference could be due to addition of coconut oil to cafeteria diet in the present study, whereas in other studies, coconut oil was administrated with standard rat chow. The present findings, however, agree with those of ***Pronczuk et al. (1991),*** who reported increased total cholesterol upon coconut oil feeding. Fortunately the results encountered in the present study,

demonstrated that the increase in plasma cholesterol was mainly in the beneficial HDL-C fraction rather than the LDL-C fraction which agree with ***Nevin and Rajamohan (2004).***

It could, thus, be concluded that administration of hypercaloric hyperlipidemic diet early in life resulted in increased body adiposity with reduced rate of increase in body length, and altered platelet function leading to enhanced platelet aggregation. Coconut oil supplementation in diet induced significant decrease in body weight and adiposity, as well as platelet aggregation. These favorable effects were obtained after 9 weeks of starting coconut oil supplementation despite continued cafeteria diet feeding. These findings make coconut oil feeding a recommended tool of weight lowering to avoid the suffering of long term dietary restriction. It could also help in alleviating the platelet dysfunctions encountered in obese individuals.

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Abbreviations:
APTT *(Activated partial thromboplastin time)*, **BMI** *(Body mass index)*, **BW** *(Body weight)*, **Caf** *(Cafeteria diet-fed*), **Caf/coco** *(Cafeteria/coconut oil diet-fed)*, **DIT** *(Diet induced thermogenesis)*, **FDP** *(Fibrin degradation products)*,**GH** (*Growth hormone)*, **HDL** *(High density lipoproteins)* , **LCT** *(Long chain triglycerides)*, **LDL** *(Low density lipoproteins)*. **MCT** *(Medium chain triglycerides)*, **PT** *(Prothrombin time)*, **TC** *(Total cholesterol)*,**TG** *(Triglycerides)*

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الملخص العربي

تأثير إعطاء زيت جوز الهند على بعض التغيرات فى ميكانيكية وقف النزف المصاحبة للسمنة فى الفئران

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اجرى هذا البحث لدراسة التغيرات التى تحدث في ميكانيكية وقف النزف المصاحبة للسمنة, و محاولة كشف اذا كانت هناك استفادة من استخدام زيت جوز الهند في احداث تحسن في هذه التغيرات.

و قد اجريت هذه الدراسة على 44 من فئران التجارب البيضاء, الذين تم تقسيمهم الى المجموعات التاليه: مجموعة الفئران الضابطة, وعددها 13 فأرا, و تمت تغذيتها بغذاء الفئران العادى طوال فترة البحث. المجموعة الثانية مكونة من فئران تم تغذيتها بغذاء عالي السعرات بداية من الاسبوع الثانى بعد الولادة ولمدة اربعة شهور لاحداث سمنة بها, ثم تم تقسيمها الى مجموعتين فرعيتين: مجموعة استمرت تغذيتها بغذاء عالي السعرات, و عددها 16 فأرا, حتى نهاية فترة البحث و المجموعة الأخرى, و عددها 15 فأرا, اضيف زيت جوز الهند للغذاء عالي السعرات فى غذائها ابتداء من الاسبوع ال15 و حتى نهاية فترة البحث ايضا.

و قد تم فى جميع المجموعات تعيين مؤشر كتلة الجسم و ايضا زمن بروثرومبين و زمن ثرومبوبلاستين الجزئي وتجمع الصفائح الدموية ومستوى نواتج تكسير الفيبرين بالاضافة الى تقدير مستوى دهون البلازما و عدد خلايا الدم المختلفة.

و قد اظهرت النتائج ان الفئران التى تمت تغذيتها بالغذاء عالى السعرات زاد موشركتلة الجسم فيها زيادة ذات دلالة احصائية مقارنة بالمجموعة الضابطة , بينما نقص مؤشر كتلة الجسم بدرجة ذات دلالة احصائية بعد اضافة زيت جوز الهند الى غذاء هذة الفئران لمدة 9 اسابيع و ذلك بالمقارنة مع المجموعة المغذاة بغذاء عالى السعرات.

و بالنسبة للتغيرات فى ميكانيكية وقف النزف, لم تكن هناك تغيرات ذات دلالة احصائية في قياسات تجلط الدم (زمن البروثرومبين- زمن الثرومبوبلاستين الجزئي- نواتج تكسير الفيبرين) فى الفئران المغذاة بغذاء عالى السعرات باضافة او بدون اضافة زيت جوز الهند, مقارنة بالمجموعة الضابطة, بينما زاد تجمع الصفائح الدموية فى الفئران المغذاة بالغذاء عالى السعرات زيادة ذات دلالة احصائية مقارنة بالمجموعة الضابطة و قد ادى اضافة زيت جوز الهند للغذاء الى نقص ذو دلالة احصائية في تجمع الصفائح الدموية مقارنة بالمجموعتين الاخرتين.

لم يحدث اى تغيير ذو دلالة احصائية بين المجموعات الثلاثة فى مستوى الدهون الثلاثية و الدهون منخفضة الكثافة, بينما زاد مستوى الكوليستيرول الكلى و الدهون عالية الكثافة فى المجموعة التى تتغذى بغذاء عالى السعرات مضافا الية زيت جوز الهند بدرجة ذات دلالة احصائية مقارنة بالمجموعة المغذاة بغذاء عالى السعرات.

و بهذا فإن نتائج هذة الدراسة تؤكد ان السمنة تؤثر سلبيا على ميكانيكية وقف النزف, بالتأثير على وظائف الصفائح الدموية بزيادة تجمعها, و ان اضافة زيت جوز الهند الى الغذاء يقلل هذا التأثير. و بهذا قد يكون اضافة زيت جوز الهند الى الغذاء فائدة فى تقليل الوزن الزائد و ايضا فى تحسن وظائف الصفائح الدموية.